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Disease-a-Month

Interactions of Pregnancy and Heart Disease

JAMES METCALFE
C. SIDNEY BURWELL

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Interactions of Pregnancy and Heart Disease

JAMES METCALFE
C. SIDNEY BURWELL

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James Metcalfe

is Assistant Professor of Medicine, Harvard Medical School, Associate Physician, Boston Lying-in Hospital and Associate Physician, Peter Bent Brigham Hospital. He received his M.D. degree from Harvard University in 1946 and his house staff training at the Peter Bent Brigham Hospital. Since 1953, Dr. Metcalfe has been an Established Investigator of the American Heart Association. His interests lie in the study of the modifications of maternal physiology during pregnancy and the effects of such modifications on the course of disease.

C. Sidney Burwell

is Samuel A. Levine Professor of Medicine Emeritus, Harvard University, Physician Emeritus at the Peter Bent Brigham Hospital and Consultant in Medicine at the Boston Lying-in Hospital. He received his M.D. degree from Harvard in 1919 and subsequent training at the Massachusetts General Hospital and the Johns Hopkins Hospital. From 1928 to 1935 he was Professor of Medicine at Vanderbilt University and from 1936 to 1949 he was Dean of the Faculty of Medicine at Harvard.

CHANGES IN CARDIOVASCULAR PHYSIOLOGY DURING PREGNANCY

A COMPLEX SERIES of integrated changes in maternal homeostasis accompanies mammalian pregnancy. These changes, which, as we understand them, appear designed for the survival of the race, are necessarily consistent with the continued survival and well-being of the mother. In the woman handicapped by disease, however, such adjustments may alter the natural history of the disease so that maternal (and thus fetal) life is endangered. This has been the historical experience with women who enter

pregnancy with damaged hearts. Knowledge of the physiology of pregnancy explains the effects of pregnancy on the physiology of heart disease. It can be demonstrated (8) that programs of management based on knowledge of this interaction of pregnancy and heart disease have been successful in lowering the morbidity and mortality of the mother with heart disease during pregnancy and in decreasing fetal mortality. Some of the known maternal adjustments during pregnancy deserve emphasis here.

Heart Rate

During pregnancy, in the normal human, the resting heart rate increases progressively to about 10 extra beats per minute (10). The increase reaches its peak at about the 8th lunar month, then declines toward the nonpregnant level in the final few weeks of pregnancy.

During labor, changes in heart rate occur with alarming rapidity (8). The significance of these changes is diminished by the fact that they are intermittent, but at the height of a contraction Adams and Alexander (2) found the heart rate to average 98 per minute as compared with 86 per minute between contractions. Postpartum, the resting heart rate falls to normal levels within a few days.

Blood Volume

The changes in blood volume during pregnancy in the normal mother (4, 30) follow essentially the same pattern as that described for changes in heart rate. Total blood volume increases by about 30% reaching this maximum at about the 30th week of pregnancy. In the last weeks of pregnancy, maternal blood volume, like maternal heart rate, declines significantly toward the nonpregnant level. The total blood volume is composed of red cell volume and plasma volume, and the major change occurs in the volume of plasma within the maternal circulation. Changes in red cell volume are not as pronounced (4). As a result, there is a progressive hemodilution until the 30th week of pregnancy despite the fact that the volume of circulating red blood cells is increased. In the final weeks, as the plasma volume falls, the hemoglobin level of circulating blood rises (27). The hemodilution of pregnancy is also shown by a decline in plasma protein

concentration (27) which reaches its lowest point approximately 10 weeks before term in the normal human.

At the time of delivery there is a further sudden decrease in circulating blood volume (26) which brings the total blood volume to within normal, nonpregnant levels within 7 days postpartum (45), although red cell volume (4) and hemoglobin concentrations (45), which fall below normal values at delivery, subsequently rise over a period of many weeks.

Cardiac Output

The output of the mother's heart increases during pregnancy, also reaching its maximum value 8–10 weeks before term. Beyond that point, as term approaches, there is a significant decline in the amount of blood pumped each minute by the maternal heart. The peak increase in resting cardiac output amounted to approximately 40% above normal in one study performed by cardiac catheterization (3).

During the uterine contractions of labor, an increase in cardiac output averaging 20%, when compared with values obtained between contractions, was found by Adams and Alexander (2). In the first few postpartum days, an elevated cardiac output persisted, although heart rate had returned to normal.

The distribution of the increased cardiac output during pregnancy is under active investigation. Munnell and Taylor (35) investigated hepatic blood flow during pregnancy and could find no significant change, McCall (29) found no change in cerebral blood flow during gestation, but Bucht (6) and, more recently, Sims and Krantz (41) demonstrated that the renal blood flow rises significantly during early and mid-pregnancy, declines to nonpregnant levels in the last trimester and in the postpartum period is decreased to levels below control observations. The increased renal blood flow during pregnancy has been attributed to endocrine factors (41). Blood flow to the hands has been shown to increase during pregnancy (1, 7), the metabolically active fetus losing heat through the active peripheral circulation of the mother. Uterine blood flow has remained an unstudied problem until recently despite its clear importance, but appears to reach levels of about 500 ml./min. at term (31). As originally suggested

by Burwell and co-workers (10) in 1938, the pregnant uterus does represent an area of lowered vascular resistance in the maternal circulation. However, it is doubtful, from studies in other species (32), that the decline in cardiac output in the last weeks of pregnancy is due to a decrease in uterine blood flow. A more likely explanation is that the decrease in renal blood flow as term approaches is shared by other viscera and masks an ever-increasing supply of maternal blood to the pregnant uterus.

The mechanisms by which these changes in blood volume, cardiac output, organ blood flow and pulse rate are brought about are unclear. Some are probably secondary to the creation of an area of low vascular resistance in the uterus during pregnancy, a hemodynamic arteriovenous fistula. Others may be due to the effects of the altered endocrine balance of pregnancy.

Other changes in the maternal circulatory apparatus accompanying pregnancy, such as the occurrence of vascular spiders and hyperemia of the palms, are less well understood but may be no less important to our understanding of the mechanisms by which pregnancy endangers the woman with heart disease.

Changes also occur in the respiration of pregnant women which are important in the diagnosis and evaluation of heart disease. The maternal oxygen consumption increases until term, when it reaches levels about 15% above that seen in nonpregnant women under the same basal conditions (10). One would expect that this increased oxygen consumption would be accompanied by proportionate increase in minute ventilation on the part of the pregnant woman since the fetus "breathes through its mother's circulation." It has been found, however, that the ventilation of the pregnant woman increases sooner and in greater proportion than her oxygen consumption; to say it another way, the pregnant woman shows alveolar hyperventilation, the partial pressure of carbon dioxide in her alveolar air falling from nonpregnant levels of approximately 40 mm. Hg to about 30 mm. during pregnancy. This hyperventilation appears to begin at the time of the last menstrual period and increases progressively as pregnancy advances so that arterial pCO_2 reaches a minimum during the last trimester (5). This maternal hyperventilation has been attributed to the action of progesterone on the respiratory centers. It results in evident benefit to the fetus since it permits a partial pressure of carbon dioxide in his blood similar to that which he will ex-

perience in extra-uterine life. Lung volumes of pregnant women have been studied repeatedly. Despite the enlarging uterine mass, there is general agreement that no significant decrease in vital capacity occurs during pregnancy. For the physician following a patient with heart disease through pregnancy this means that a decrease in this easily measured function has the same significance as when it is seen in the nonpregnant patient.

Several definite limitations to the studies of circulatory and respiratory physiology during pregnancy should be noted. Most of the studies referred to have involved only *normal* pregnant women in the *resting* state. It must be demonstrated that they occur in pregnant women with heart disease and the studies must be extended to the usual conditions of active life characteristic of young women.

Observations by Schmidt (37), Strayhorn (42), Werkö (47) and Cornett (12), although not definitive, document an increase in cardiac output during pregnancy in women with several varieties of heart disease. In our laboratory (8), it has been demonstrated that the changes in respiration and lung volume which accompany normal pregnancy are also seen during gestation in women with heart disease.

Muscular exercise during pregnancy will be more costly in terms of oxygen consumption because of the weight gain characteristic of pregnancy. Moreover, the studies of Widlund (48) suggest that, at least in severe exercise, there is a reduction of muscular efficiency during pregnancy. Further work is needed to clarify this area and to analyze its importance in women with heart disease. Werkö (47) and Bader and co-workers (3) could demonstrate no change in the response of cardiac output to exercise during pregnancy, but their studies are not conclusive. Our opinion, based on inadequate evidence (8), is that exercise during pregnancy carries a higher cardiac cost than when it is performed in the nonpregnant state.

In summary, increases in cardiac output, heart rate and blood volume of significant magnitude occur during pregnancy in normal women and in women with heart disease. The change in each of these reaches its maximum not at term, but several weeks before term. During labor, rises of heart rate and cardiac output have been demonstrated but at that time are intermittent. In the postpartum period, pulse rate and blood volume return rapidly

to normal values and red cell volume is reduced but cardiac output remains above nonpregnant values well into the postpartum period.

DIAGNOSIS OF HEART DISEASE IN PREGNANT WOMEN

The accurate diagnosis of heart disease is made more difficult by the hemodynamic changes which accompany pregnancy. A variety of murmurs may develop and may be incorrectly attributed to valvular deformity. Approximately 50% of the patients referred to the Boston Lying-in Hospital Medical Clinic with a murmur believed by the obstetricians to be suggestive of heart disease are found in the postpartum period to have no clear evidence of organic heart lesions. In general, such murmurs are systolic in time and of low or medium intensity. Occasionally, in the presence of thyrotoxicosis, anemia or fever complicating pregnancy, they are of an intensity usually significant of heart disease. Hurst and co-workers (23) have emphasized the importance of murmurs arising in the anterior chest wall and breast in the last few months of pregnancy. Scott and Murphy (39) have reported 2 patients in whom a continuous murmur which was heard late in pregnancy disappeared after lactation. These murmurs were best heard in the parasternal area and disappeared when firm pressure was applied to the anterior chest wall with a stethoscope. A third heart sound occurs with increased frequency during pregnancy in normal women (15). On the other hand, the auscultatory evidence of established heart disease, such as the rumbling diastolic murmur of mitral stenosis, may be reduced in intensity by the tachycardia of pregnancy.

Enlargement of the cardiac shadow by x-ray and minor electrocardiographic changes are frequently seen without accompanying evidence of heart disease and are apparently due to displacement and rotation of the heart due to the high diaphragm.

Occasionally, the hyperventilation which accompanies pregnancy may be accompanied by dyspnea and in a few instances hemoptysis occurs during pregnancy without other evidence of heart or pulmonary disease. Palpitation is common and the occurrence of paroxysmal tachycardia has been shown to be increased (44).

In general, an original diagnosis of organic heart disease should be made with more reluctance during pregnancy than in the nonpregnant state. In many cases, final evaluation must await the postpartum period.

The incidence of heart disease in women of the child-bearing age will vary with geographic location, with the availability of diagnostic facilities, with the interest and level of suspicion of the observers and with the frequency of such predisposing factors in the hospital population as nutritional deficiency, toxemia of pregnancy and hypertensive heart disease. At the Boston Lying-in Hospital, the incidence of organic heart disease has varied between 1 and 3% of the patient population for many years. Eighty-five per cent of our cardiac patients have rheumatic heart disease. The remainder nearly all have congenital deformities of the heart or great vessels. Heart disease due to hypertension is rare in a predominantly white female population of child-bearing age, and coronary artery disease is even rarer. Thus the major part of our concern is with the pregnant patients with rheumatic heart disease. Of these, most have mitral stenosis as their predominant or only valve lesion.

It has been demonstrated adequately and repeatedly in the past that pregnancy represents a time of increased hazard to the life and health of the woman with heart disease (8). Recent reduction of maternal mortality to levels of 1% should not be regarded as evidence of the lack of danger to such patients but as evidence that by careful management the danger can be minimized. Successful management must be based on a clear understanding of the mechanisms by which the physiologic changes of pregnancy threaten the life of the woman with a diseased heart.

The changes in cardiac output and plasma volume, pulse rate and respiration which accompany pregnancy are not in themselves dangerous. They appear to occur to some degree in all pregnant patients but their sinister implications are limited to those patients with organic cardiac deformities.

The most important and clearest example of the mechanism by which the physiologic changes accompanying pregnancy jeopardize the health and lives of patients with heart disease can be drawn around the physiology of mitral stenosis.

RHEUMATIC HEART DISEASE

Mitral Stenosis

The fundamental hemodynamic problem in patients with mitral stenosis is presented by the narrowed orifice at the mitral valve, which obstructs blood flow from the left atrium to the left ventricle during diastole. As a result of this obstructive lesion to left atrial outflow, the pressure in the left atrium mounts. The height of rise of left atrial pressure depends on the cardiac output, the time available for flow from left atrium to left ventricle (that is, the diastolic time per minute) and the volume of blood contained within the left atrium and its tributary pulmonary veins. An increase in cardiac output can only occur in such a situation by elevation of left atrial pressure. An increase in heart rate decreases the time in each minute available for flow from left atrium to left ventricle and, in the patient with severe mitral stenosis, tachycardia of any origin will cause a further rise in left atrial pressure.

An increase of blood volume in a pulmonary vascular bed that is already distended with blood will cause further distention and a further increase in pressure. Left atrial hypertension is necessarily accompanied by pressure increases of similar magnitude in the pulmonary veins and pulmonary capillaries. Furthermore, secondary changes in the pulmonary arterioles act to cause pulmonary arterial hypertension and eventual right heart failure in many patients. *Myocardial failure,* however, is a late event in the natural history of mitral stenosis. As pointed out by Wood (49), the usual course of events in the patient with developing mitral stenosis, once the attack of rheumatic fever is survived, includes a 20-year period without symptoms, followed by progressive disability. Most women with mitral stenosis are asymptomatic in the early years of child bearing, even without restriction of diet or activity. But even in such patients pulmonary venous and pulmonary capillary hypertension frequently can be demonstrated at rest.

Let us now consider what happens when the patient with mitral stenosis becomes pregnant. As pregnancy advances, blood volume, cardiac output and pulse rate rise. These three well-documented changes all work toward further elevation of the left atrial, pulmonary venous and pulmonary capillary pressures. In a patient

with mitral stenosis, an increased cardiac output can be accomplished only by further elevation of pressure in the left atrium. Furthermore, blood flows from the left atrium to the left ventricle only during diastole. With an increased heart rate, the diastolic time per minute is shortened and the *velocity* of blood flow through the stenotic mitral valve must therefore be increased if cardiac output is to be maintained. Again, this is possible only by further elevation of left atrial pressure. The increased blood volume during pregnancy appears to be shared between the pulmonary vascular and peripheral vascular circuits. In a pulmonary capillary and venous bed which is already distended, the increased blood volume of pregnancy accentuates the hypertension in these fragile and important vessels. Hypertension in the pulmonary venous system may cause rupture of a bronchial or pulmonary vein with hemoptysis; usually this is a relatively unimportant event (49) but occasionally causes well-justified alarm (18). Hypertension in the pulmonary capillaries when it reaches levels over 30 mm. Hg leads to transudation of fluid into the alveolar walls and alveoli with resultant dyspnea, orthopnea and, if sustained, pulmonary edema.

These, we believe, are the main mechanisms by which pregnancy threatens the health of the woman with mitral stenosis. The degree of pulmonary capillary and venous hypertension at any moment will depend on the severity of mitral stenosis in that particular patient, the cardiac output demanded of the patient's heart and the diastolic time per minute available for flow. As the cardiac output, blood volume and heart rate all rise to their maximum values at about the 30th week of pregnancy, pulmonary capillary pressure increases concomitantly. In the final weeks of pregnancy, as cardiac output, blood volume and heart rate decline toward their normal levels, pulmonary capillary pressure similarly declines and the danger of transudation and pulmonary edema lessens. Hamilton and Thomson (22) demonstrated from their extensive experience that the peak incidence of pulmonary edema during pregnancy in patients with heart disease occurs at the point of maximum cardiac output, blood volume and pulse rate.

It was the experience of Hamilton and Thomson that, although the peak incidence of pulmonary edema came at the end of the 8th lunar month, 75% of all deaths in cardiac patients occurred

in the puerperium. Recent studies (2) have shown that the cardiac demands of pregnancy are continued, although diminished from the pregnancy peak, in the postpartum period.

It should be emphasized at this point, however, that pregnancy is only one of the many factors which may operate to increase left atrial and pulmonary vascular pressures. Exercise, anemia, obesity, infections with accompanying fever all act on these important pressures in the same direction as pregnancy does. Pregnancy is not a *unique* danger to the patient with mitral stenosis; and pregnancy is seldom an *isolated* danger. If the pregnant patient with mitral stenosis develops one or several of these other disturbances which tend to increase her cardiac burden in the same direction, the *sum total* of burdens may be intolerable, whereas the burden of pregnancy itself is within the capacity of her heart.

In considering the management of a patient with mitral stenosis during pregnancy, three possibilities seem to exist. These are not mutually exclusive; we believe that each still has its place in the successful planning for life and health of the cardiac patient. The first of these possibilities is the denial, prevention or interruption of pregnancy. This approach, which has been used frequently in the past, is based on the concept that by prevention of the burden of pregnancy the longevity and health of the woman is prolonged. Although it is the unanimous opinion of most observers today that interruption of pregnancy is itself not without hazard, realistic family planning still seems to us important in the lifelong management of women with heart disease. The second basic concept is that of careful medical management before, during and after pregnancy; such management is indispensable for optimum results. The patient with heart disease should be in optimum physical condition, including cardiac condition within the limits of her heart disease, before pregnancy is undertaken. She should preferably be past the age of high incidence of recurrent rheumatic fever. She should not be anemic or obese. She should have been carefully evaluated for the advisability of cardiac surgery and, if indicated, this should be performed before pregnancy is undertaken.

During pregnancy, medical management is based on the concept of limitation of the *total* burden on the diseased heart. The fundamental concept is that pregnancy is only one element in a

shifting complex of loads, each of which must be considered in managing the total cardiac economy. Factors such as activity, anxiety, and such disease states as thyrotoxicosis, anemia, obesity and infections must be sought for and, whenever possible, removed so that a place is made in the total cardiac budget for the temporary expenditures of pregnancy. Furthermore, the cardiac burden of pregnancy itself, as expressed by changes in blood volume, cardiac output and pulse rate can be demonstrably reduced by sodium restriction (8). The possibility of recurrent rheumatic fever or subacute bacterial endocarditis must be guarded against. The intensity of limitation of activity and sodium restriction are regulated according to the needs of the specific patient and according to the known alterations during pregnancy in the intensity of the cardiac load. It cannot be emphasized too strongly that such medical care is necessary even in patients who have undergone successful cardiac surgery for acquired heart disease and who are brought by this and other measures into pregnancy with the best possible cardiac ability. Furthermore, the value and necessity for medical planning and management does not end with pregnancy. Successful tolerance of the long years of motherhood demands that the cardiologist re-evaluate regularly the changing burdens on the heart of the patient with an eye to proper re-emphasis of medical and surgical procedures.

Critical evaluation of the place of surgery in the management of pregnant cardiac patients is difficult at present. Whenever possible, evaluation of the cardiac status before pregnancy should have been carried out and the advisability of valve repair judged with regard to the known burden of the coming pregnancy. If, under optimum medical care, symptoms of pulmonary congestion persist, valvotomy should be performed before final evaluation for possible pregnancy. If this opportunity is not taken, and the patient presents herself with symptoms once pregnancy has begun, the decision is more difficult. The current surgical procedures designed for relief of acquired valve disease are never curative and their application to the individual patient demands a depth of experience which is difficult to apply in the complex situation of pregnancy. Certainly mitral valvotomy can be performed on the pregnant woman with heart disease (16, 21, 46).

Certainly also this is unnecessary in the great majority of patients with mitral stenosis who successfuly negotiate the hazards of pregnancy with a mortality of approximately 1% on medical therapy alone. The current safety and success of mitral valvotomy have led us to change our previous views (9) slightly. If, on good medical management, symptoms and signs of pulmonary congestion appear early in pregnancy, we now believe that mitral valvotomy may be better than interruption of pregnancy. The following reservations persist: (1) The surgeon must be able to perform as good a valvotomy as if pregnancy were not present, because re-operation is hazardous and difficult. (2) The physician must recognize that his responsibilities do not end with the decision for surgery. All patients will need careful medical care after valvotomy, even when the dangers of the operative and immediate postoperative periods have passed. (3) The patient must recognize that however good the operative result she will need continued counsel and care. Most patients retain some symptoms, all need prophylaxis against recurrent rheumatic fever and subacute bacterial endocarditis. With these reservations, and with careful medical management and surgical evaluation, we have still not advised valvotomy during pregnancy, but are prepared to do so. The need is small but important to an occasional patient.

If symptoms and signs of pulmonary congestion appear after the 4th month of pregnancy, the hazards of surgery appear prohibitive. We advise weathering the pregnancy by a rigid medical regimen under hospital conditions. The tendency to "do something" is understandable, but the burden of pregnancy has now become a major factor and is better not complicated by surgical and anesthetic burdens (14) whether designed to interrupt pregnancy or to correct valve deformity.

Other Forms of Rheumatic Heart Disease

As stated previously, mitral stenosis is the most common and the most important form of valvular heart disease encountered during pregnancy. When other rheumatic valvular lesions predominate, the danger of pulmonary congestion during pregnancy is less than in the patient with mitral stenosis. When the main

heart lesion is mitral stenosis, the cause of pulmonary capillary hypertension and pulmonary edema is the mechanical obstruction offered by the deformed valve. In mitral regurgitation or lesions of the aortic valve, sustained pulmonary hypertension awaits the onset of left ventricular failure. This is an uncommon development in the child-bearing age. In general, then, we are less frequently and less deeply concerned about the dangers of pregnancy in patients with predominant mitral regurgitation or aortic valve disease than in patients with predominant mitral stenosis.

A word of conservatism about the significance of isolated systolic murmurs during pregnancy: Since systolic murmurs occur in fully 50% of normal hearts under the hemodynamic stress of child bearing, it is frequently necessary to wait for postpartum re-evaluation before a firm diagnosis can be made. As mentioned above, the importance of making such a diagnosis is less urgent than is the case with mitral stenosis. But patients with pure mitral regurgitation do occur and such patients should be recognized, especially for prophylaxis against subacute bacterial endocarditis.

Care during pregnancy for patients with mitral regurgitation as their sole or predominant valve lesion is largely a matter of watchful waiting. Pulmonary congestion is rare, but it should be sought for by careful questioning, weighing, auscultation and determination of the vital capacity on each visit. If evidence of left ventricular failure does appear, immediate hospitalization, sodium restriction and diuresis and digitalization are indicated. Surgical measures for the correction of mitral regurgitation are not yet acceptable.

Congestive heart failure on the basis of aortic stenosis is rare in the child-bearing age. This lesion, however, predisposes to conduction disturbances and syncopal episodes. During pregnancy, many women have evidence of myocardial irritability, and fainting is common. Management of the pregnant patient with aortic stenosis must include warnings against sudden exertion, continued fatigue or sudden assumption of the upright posture. We avoid restriction of sodium intake in such patients because the danger of pulmonary congestion is less than the danger of syncope, which may be fatal. At each clinic visit, however, evidence of pul-

monary congestion must be sought. Its occurrence would be ominous, necessitating immediate hospitalization and vigorous therapy. The surgical treatment of aortic stenosis is now becoming an acceptable procedure. It should not yet be advised for asymptomatic patients (49). We would avoid it during pregnancy, but it is taking its place in the long-term management of patients with this lesion.

Patients with important degrees of aortic regurgitation comprise about 10% of our experience with pregnant cardiac patients, an incidence greater than that of "pure" mitral regurgitation and much greater than that of predominant aortic stenosis. These women, with large forceful cardiac beats, visible collapsing carotid pulsations and alarming murmurs, are watched carefully during pregnancy, but we have not observed the occurrence of pulmonary congestion. Because two of them have experienced sudden vascular collapse at delivery (8) we avoid sodium restriction during pregnancy and encourage venous return immediately following delivery, using such simple measures as elastic stockings, elevation of the foot of the bed and avoidance of the flaccid supine position.

Tricuspid and pulmonary valve disease on a rheumatic basis have not been encountered as important lesions in our experience with pregnant cardiac patients.

RHEUMATIC FEVER: SUBACUTE BACTERIAL ENDOCARDITIS

Two further matters for discussion in connection with rheumatic heart disease are active rheumatic fever and subacute bacterial endocarditis. Patients with a past history of rheumatic fever, whether or not heart disease has resulted, need protection against recurrence of this disease. Such protection is afforded by the continuous administration of penicillin, or, in penicillin-sensitive patients, sulfadiazine (33). Although the occurrence of recognizable rheumatic fever is rare in pregnancy (36), its prognosis in the few reported cases has been poor. We recommend immediate institution of prophylaxis in all patients with rheumatic heart disease or a clear history of rheumatic fever at the time of their first visit. Since most of our patients are pregnant when we first see them, prophylaxis is begun during pregnancy. Indeed,

it needs noting that the occurrence of pregnancy is still, in our civilization, the usual reason for a woman's first contact as an adult with the medical profession. The opportunity, and attendant responsibility, of the obstetrician has not been sufficiently recognized in the past.

Subacute bacterial endocarditis has become less important with the use of prophylactic measures. All patients with congenital cardiac deformities or acquired valve disease should be instructed in their need for antibiotic protection at times of bacteremia due to dental manipulation or genitourinary surgery. Delivery, by cesarean section or vaginally, has been regarded as a time and cause for bacteremia, although evidence for this is lacking in normal women without genital infection. The dangers of increased penicillin dosage in women with heart disease, most of whom are using penicillin regularly for prophylaxis against rheumatic fever, are so small and the implications of bacterial endocarditis are so bad, that we routinely advise large doses of penicillin in such patients beginning with the onset of labor and continuing for 3 days into the postpartum period (8). We have not seen a case of subacute bacterial endocarditis following delivery.

CONGENITAL HEART DISEASE

Because of recent developments in the diagnosis and correction of congenital cardiac deformities, this group of patients has become numerically significant in the population entering pregnancy. It is to be hoped that as corrective surgical procedures are developed, these patients will be detected during childhood and that surgery will be carried out before the child-bearing age. But, because of the present low incidence of routine health examinations in much of our population, some women still enter pregnancy with undiagnosed congenital heart disease. Again, the obstetrician must have a sufficiently high level of diagnostic suspicion to direct such patients toward diagnosis. The lesions to be looked for are those consistent with life to the child-bearing period. These include such obstructive lesions as coarctation of the aorta and pulmonary stenosis, the congenital arteriovenous fistulas caused by persistent patency of the ductus arteriosus and defects of the cardiac septa, and such combined lesions as the

tetralogy of Fallot and the Eisenmenger complex. A few words about each of these lesions will serve to summarize the small experience which we have had with it during pregnancy.

Pulmonary Stenosis without a Septal Defect

Pure pulmonary stenosis on a congenital basis is not rare. The obstruction may be at the pulmonary valve or in the infundibular area of the right ventricle. The right ventricular output is impeded and, after birth, when the volume of blood flow through the lungs increases, normal flow can be accomplished only by an increased right ventricular systolic pressure. Such a handicap, however, is frequently compatible with survival to the childbearing age. Such patients may present themselves during pregnancy without symptoms. We have reported 7 patients (8) with the diagnosis of pure pulmonary stenosis who have delivered a total of 7 babies. They were essentially asymptomatic before and during pregnancy and were managed by the conservative principles detailed previously. It should be realized that a relatively low cardiac output is often associated with pure pulmonary stenosis and that syncope may occur during or after pregnancy. Postpartum collapse may occur.

An important function of the obstetrician and his consultant cardiologist should be to insure thorough investigation of the lesions and evaluation for surgery (20) as part of the long-term planning for these patients.

Primary Pulmonary Hypertension

Another obstructing lesion to the outflow of blood from the right ventricle, is due to an increased vascular resistance in the pulmonary arterioles. This is called "primary" pulmonary hypertension and appears to be important in pregnant patients because of a tendency to sudden syncope, which may cause death. Such a catastrophe has been reported by Jewett and Ober (24). Such circulatory collapse may appear at the time of delivery or in the first few hours postpartum. It appears to be on the same basis as the circulatory collapse associated with pulmonary stenosis and should be treated by leg bandages, abdominal binders and other methods of encouraging venous return. In such patients, sodium

should not be severely restricted unless clear evidence of circulatory congestion is present.

Coarctation of the Aorta

Coarctation of the aorta is predominantly a disease of males. It is often associated with other abnormalities of the heart or vascular system. It is consistent with a normal development to the child-bearing age in many female patients. The diagnosis should be considered in every patient with systolic hypertension. In most patients with this disorder, the diagnosis can be made by routine examinations that are permissible during pregnancy. Unless symptoms are present, repair of the coarctation during pregnancy should not be considered. Strayhorn (42) demonstrated an increased cardiac output in one patient with coarctation during pregnancy as compared with her own values in the postpartum period. This increased cardiac output was *not* accompanied by an increased hypertension in the patient's arterial system. The burden of an increased cardiac output in pregnancy was not compounded by a simultaneous increase in arterial blood pressure.

One of the dangers facing patients with coarctation of the aorta is arterial rupture. This may occur in the form of a dissecting aneurysm, usually in the ascending aorta, or aneurysms associated with collateral vessels may rupture. The dangers of dissecting aneurysm of the aorta during pregnancy and the postpartum period have been emphasized by Schnitker and Bayer (38). It does appear that the possibility of arterial rupture is increased during pregnancy particularly in patients with coarctation of the aorta, although the danger of arterial rupture is not confined to such patients (8). The basis of this danger does *not* appear to be increased intra-arterial pressure; structural alterations in the arterial walls during pregnancy have been postulated as an explanation. The dangers of pregnancy to patients with aortic coarctation have been overemphasized. Shanahan and coworkers (40) showed from their review of the literature that rupture of the aorta is more likely to occur during the final month of pregnancy than during labor, and concluded that the rarity of this occurrence during or immediately after labor did

not support the contention of previous observers who felt that the presence of coarctation was an indication for cesarean section. The danger of subacute bacterial endocarditis may be obviated by the use of antibiotic prophylaxis at delivery.

During pregnancy, then, these patients should be given a correct diagnosis. Activity, weight, hemoglobin level, sodium intake and all the other factors which may burden the heart should be assessed and controlled. Cesarean section does not appear to be indicated by the existence of coarctation but, after pregnancy is complete, evaluation of the patient for repair of the congenital defect is a responsibility of the obstetrician and his consultant cardiologist.

We have reported (8) an incidence of congenital heart disease of 3.6% in living offspring of mothers with coarctation of the aorta. This is approximately 10 times the incidence in a normal population and emphasizes the genetic factor in the occurrence of congenital heart disease.

Patent Ductus Arteriosus

Patency of the ductus arteriosus is more common in females than in males and is compatible with survival to adult age. The presence of a patent ductus is adequate indication for its repair except when it serves as a pathway for accessory pulmonary blood flow in some complicated congenital defect; operation should be carried out before the development of secondary pulmonary hypertension. Surgery preferably should be carried out in childhood but in most patients who reach adult life operation is not to be regarded as an emergency procedure and we think operation should be delayed until pregnancy is completed. Occasionally, a patient exhibits evidence of myocardial failure during pregnancy. If this occurs in the first months, surgical correction may be advisable. After that time, conservative management appears safer. These patients should be guarded from subacute bacterial endocarditis, and the danger of sudden hypotension at delivery should be appreciated. Such vascular collapse, which appears to be due to sudden reduction in venous return, may, in these patients, lead to reversal of blood flow through the ductus. The possibility of the syndrome should be recognized so that its occurrence can be

met with measures designed to maintain peripheral blood pressure and increased venous return.

Atrial and Ventricular Septal Defects

Women with defects of the atrial or ventricular septum frequently live through the child-bearing age. In general, they do well in pregnancy. However, the danger of arterial hypotension and circulatory collapse is present in these patients at delivery and may be accompanied by reversal of the shunt flow. The danger should be recognized; if the syndrome develops, it should be combated immediately by measures designed to encourage venous return and maintain arterial blood pressure.

Tetralogy of Fallot

This complex of lesions is consistent with life to the child-bearing age. Because of the relative rarity of these patients in a pregnant population the suggestion has been made (8) that a lowered fertility is characteristic of these cyanotic persons. During delivery, and in the immediate postpartum period, episodes of syncope and increased cyanosis are sometimes seen and death has been reported. Measures designed to increase venous return and sustain blood volume are advised at the time of delivery. When evidence of myocardial failure exists, pregnancy is contraindicated, at least until surgical evaluation can be completed. If myocardial failure occurs during pregnancy, the use of medical measures seems advisable rather than the complicated surgical technics necessary to relieve the tetralogy of Fallot.

Other Congenital Heart Lesions

Other lesions such as the Eisenmenger complex and tricuspid atresia with an atrial septal defect may be consistent with survival to and through pregnancy and the production of healthy children (1).

It is beyond the scope of this article to discuss surgical correction of congenital defects. Sometimes this can be done with minimum hazard before the patient enters the child-bearing age. This is clearly true for patency of the ductus arteriosus and for

coarctation of the aorta, before the occurrence of symptoms and signs of progressive cardiac deterioration. With other forms of congenital defect, the case is not so clearly in favor of "prophylactic" surgery in the asymptomatic child. However, recent technical and surgical advances, including the development of practical pump oxygenators for total body perfusion, make it clear that all cardiac lesions may need to be considered for repair before evidences of cardiac, vascular or general inadequacy develop (43). In the words of Friedberg (17) "This period of transition from closed to open cardiac surgical techniques imposes a special difficulty for the physician who must determine whether the desirability or even urgency for cardiac surgery is such that he must recommend the safer but less certain or less curative closed procedure or whether he should recommend delay until his surgeon can perform a preferable open cardiac operation without significant additional risk." It should be emphasized here that one of the great responsibilities of the obstetrician lies in the unique opportunity presented by the asymptomatic woman with heart disease. He must have a high index of suspicion and take the time to do a careful physical examination. He must be ready to refer patients on the basis of *suspicion* of heart disease for accurate cardiac evaluation, followed by care during pregnancy and proper orientation for careful cardiac care in the ensuing years.

In general, patients with congenital defects, when asymptomatic, tolerate pregnancy well; in the absence of symptoms, we have delayed surgical intervention until the completion of pregnancy. Although surgical procedures designed for the correction of various anomalies have been performed without apparent untoward effects during pregnancy, in our hands the policy stated above has been unassociated with maternal mortality. Part of our hesitancy to perform surgery on the heart or great vessels during pregnancy is based on the observation of increased blood vessel fragility associated with pregnancy. This is manifested by the occurrence of vascular spiders, and by an increased incidence of rupture of splenic and renal arterial aneurysms and of aortic dissection. Should symptoms of low cardiac output or pulmonary congestion develop during pregnancy in one of these patients, we would still hope to weather the storm by careful medical handling, planning corrective surgery in the postpartum period.

DISORDERS OF THE HEART BEAT

It has long been recognized that there is a tendency to certain disorders of the heart beat during pregnancy. Those most frequently encountered are: sinus tachycardia, sinus bradycardia, premature beats originating in auricles or ventricles, paroxysmal atrial tachycardia and atrial fibrillation.

Sinus tachycardia is defined as a heart rate greater than 100 beats per minute, originating in a normal sinus mechanism. It is not common during uncomplicated pregnancy. However, a relative tachycardia in the basal state has been mentioned previously as one of the normal maternal adjustments during pregnancy. Under basal conditions, the maternal heart rate is approximately 10 beats per minute quicker at the 32d week of pregnancy than it is in the nonpregnant state. With this basal tachycardia there is an increased danger of superimposed tachycardia due to anemia, infection, exertion or acute blood loss. The basal tachycardia itself may be dangerous for patients with mitral stenosis as previously mentioned, and when emphasized by the superimposed factors enumerated above the danger is increased.

Sinus bradycardia is defined as a rate of less than 50 per minute originating in a normal sinus mechanism. Bradycardia of such rates occurs in the postpartum period in an occasional patient. The mechanism of this has not been worked out but we welcome its occurrence, particularly in patients with obstructive mitral stenosis.

Atrial and ventricular premature beats are common in young women and appear to be increased in frequency during pregnancy. They must be distinguished, when frequent, from atrial fibrillation; they are not significant of heart disease. Reassurance as to the normality of the heart is all that is required in treatment, but occasionally a patient profits from quinidine sulfate if it decreases the frequency of her premature beats.

Paroxysmal atrial tachycardia frequently occurs in patients with an otherwise normal heart. When it occurs in a patient with mitral stenosis, it may cause sufficient elevation of left atrial, pulmonary venous and pulmonary capillary pressures for the development of pulmonary edema. Szekely and Snaith (44) have presented evidence that paroxysmal atrial tachycardia is more common in pregnancy than in the nonpregnant state. Six of their

10 patients had rheumatic heart disease. Our own experience consists of 4 patients with documented paroxysmal atrial tachycardia. Of these, 3 had rheumatic heart disease. In general, episodes of atrial tachycardia are well tolerated by the normal heart. When mitral stenosis coexists, however, an effort should be made to minimize the frequency of occurrence by the use of quinidine sulfate.

Atrial fibrillation is seen most frequently in patients with rheumatic mitral stenosis. In such patients, the onset of atrial fibrillation may lead to sufficient ventricular tachycardia to cause pulmonary hypertension and congestion; this is corrected by slowing the ventricular rate with digitalis. Atrial fibrillation occasionally develops in patients without valvular heart disease, sometimes on the basis of thyrotoxicosis and occasionally without any apparent precipitating factor. We have recorded one such patient seen during pregnancy (8). When atrial fibrillation occurs for the first time during pregnancy, whether on the basis of mitral stenosis or without underlying heart disease, it seems worth while to attempt reversion hoping that when pregnancy is complete recurrence of the abnormal rhythm will not take place. For this purpose, we use increasing oral doses of quinidine, as outlined by Levine (25).

We have observed one patient in the Medical Clinic of the Boston Lying-in Hospital with congenital complete heart block who has gone uneventfully through two pregnancies and is currently asymptomatic in her third pregnancy. On the basis of this experience and others reported in the literature, it appears that complete heart block on a congenital basis is no handicap to successful pregnancy. On the other hand, some patients have complete heart block on the basis of degenerative heart disease. Such patients rarely are seen in the child-bearing period but have an entirely different prognosis from those with congenital complete heart block. Mowbray and Bowley (34) collected 20 patients with acquired complete heart block during pregnancy, of whom 14 had persistent complete heart block. In this group, the maternal mortality was 20%, but the factor determining prognosis was the functional status of the heart and not the presence or degree of heart block.

HYPERTENSION AND HYPERTENSIVE HEART DISEASE

Pregnant women may exhibit all the varieties of hypertension that are seen in nonpregnant patients. They also have one variety that appears to be specific for the pregnant state—preeclampsia. The diagnosis of hypertension is made on the basis of a persistent diastolic blood pressure of 90 mm. Hg or greater. Hypertensive heart disease is diagnosed when evidence of left ventricular hypertrophy, demonstrable coronary artery disease or abnormality of cardiac function develops in a patient with persistent hypertension.

In general, hypertension due to any of its many causes is present for years before evidence of cardiac involvement appears and some patients with hypertension never develop evidence of cardiac involvement. The first symptom of the development of hypertensive heart disease is usually dyspnea on exertion, followed by orthopnea and episodes of paroxysmal dyspnea, usually nocturnal. When such evidence of pulmonary congestion appears, disability is likely to be recurrent unless there are removable factors which have been partly responsible for the development of congestive failure. This allows us to emphasize that the management of congestive heart failure *in general* is based on the concept of removable burdens much as has been developed around pregnancy. Pregnancy is only one of a shifting complex of cardiac burdens, some of which are alterable or removable.

Pregnancy is usually accompanied by an increase in cardiac output and a decrease in mean peripheral blood pressure. This diminution in peripheral resistance occurs in animals with experimental hypertension produced by restriction of renal blood flow. Some hypertensive women exhibit a similar decline in diastolic blood pressure during pregnancy. This is said to occur in about 30% of hypertensive patients. In another third, the hypertension appears to be uninfluenced by pregnancy and in the remaining third there is exaggeration of the hypertension in the latter part of pregnancy. In some of these latter patients, albuminuria appears and it is considered that preeclampsia has been superimposed on pre-existing hypertension.

In our clinic population, disability and death from cardiac mechanisms based on hypertensive disease are rare in pregnant women. The disease involves an age group most of whom are

beyond the reproductive period. When hypertension becomes exaggerated by the superimposition of preeclampsia, the evolution of hypertensive heart disease may be accelerated. In a few patients, this may lead to left ventricular failure during pregnancy. There is some evidence (11) that preeclampsia worsens the long-term prognosis of the patient with hypertensive disease.

The objective of management of patients who enter pregnancy with hypertension is the prevention of toxemia of pregnancy and other factors which tend to aggravate hypertension. Perhaps the most important controllable cause of hypertension is urinary infection. Meticulous care should be taken to prevent this and to treat it effectively when it does occur. Catheterization of the bladder should be avoided in all patients but particularly in those with hypertension. In addition to avoidance and prompt control of urinary infection, obesity should be controlled by suitable diet. Emotional factors should be investigated. In addition, one must concern himself with control of elevated blood pressure itself. Hypotensive drugs have been emphasized as a mechanism for this, but frequently other measures such as reorganization of activity and reorientation of philosophy are more important in regulating the long-term outlook. If, despite these measures, hypertension persists or worsens during pregnancy, restriction of activity, anxiety and avoidance of anemia should be used to prevent the development of left ventricular failure.

In the group of women who enter pregnancy with hypertension, maternal mortality appears to be confined almost exclusively to those patients who develop superimposed preeclampsia. Since so much can currently be done to diminish the incidence of preeclampsia, mortality in hypertensive women should be diminished.

For a number of years, the incidence of preeclampsia has been falling progressively in the clinic patients of the Boston Lying-in Hospital. Our experience with this disease has therefore been small. It appears likely that most of this diminution in incidence is due to restriction of sodium intake, but, because the mechanism of preeclampsia is poorly understood, there may also be other background influences.

All pregnant patients, particularly those with underlying hypertension, should be watched with meticulous attention to sudden changes in weight, increases in blood pressure or the development of albuminuria. At the earliest sign of development

of preeclampsia, immediate restriction of activity in the hospital and rigid sodium restriction are indicated. Under this regimen, the evidence of developing preeclampsia may disappear in most patients. In a few, hypotensive agents will be necessary to control the hypertension. When evidence of pulmonary congestion appears in such cases during pregnancy, it should be managed by immediate hospitalization and use of the familiar measures for control of cardiac failure, including digitalis and mercurial diuretics and, in the acute phase, tourniquets and phlebotomy.

Another specific hazard appears to exist during pregnancy in patients with hypertension. This complication is dissecting aneurysm of the aorta which may occur during or after pregnancy in patients with hypertension (8).

MISCELLANEOUS FORMS OF HEART DISEASE

Coronary artery disease seldom occurs in females of childbearing age. We have encountered it occasionally. Most of the reported patients with angina pectoris and most women with a documented myocardial infarction before pregnancy can survive pregnancy and produce a living child. It is clear, however, that for some such women pregnancy is a special hazard which is occasionally so great that termination of pregnancy should be recommended. Such a radical procedure, however, should be accepted only after an attempt has been made to evaluate the importance of other and possibly controllable factors, and after careful observation of the effects of pregnancy on the manifestations of coronary artery disease in the patient under consideration.

Postpartum heart disease is a rare disorder of unknown etiology whose limits are not clearly defined at present. Although it appears not to occur in our clinic population, we are convinced that such a syndrome does occur, generally in the 2 months immediately following parturition, particularly in patients who have had preeclampsia during pregnancy or a long period of inadequate nutrition. The usual general principles of care for patients with cardiac failure and, in addition, supplementary caloric and vitamin intake and prolonged restriction of physical effort are recommended.

Myocarditis due to recognized infections or of the idiopathic form may occur in association with pregnancy; the relationship

appears to be coincidental. Some of the patients described as having postpartum heart disease resemble some of the patients described as having isolated myocarditis. The prognosis of this condition is generally regarded as poor but the importance of this is limited by the narrow diagnostic tools presently available. At any rate, severe and sometimes fatal myocardial degeneration can be associated with pregnancy and the puerperal state. The character of the inflammatory changes suggests that the anti-inflammatory action of adrenal steroids might be helpful but so far they appear to have been disappointing.

Heart disease secondary to thiamin deficiency may occur as a result of the extra dietary requirements of pregnancy and lactation. An adequate dietary intake and systematic vitamin administration are indicated in all pregnant patients.

PROGNOSIS OF PATIENTS WITH RHEUMATIC HEART DISEASE DURING PREGNANCY

The results of management using the principles enumerated in the preceding pages should now be presented and evaluated. Table 1 shows that we have supervised the care of 476 pregnancies in women with heart disease. Eighty-three per cent of these pregnancies occurred in women with rheumatic heart disease and we have an experience amounting to 395 pregnancies in such women. Because of the various lesions grouped together under the heading of "Congenital Heart Disease," the significance of our experience in any one group of patients is limited. For this reason, our experience with each form of congenital lesion is given in the text. The results which will now be discussed apply entirely to patients with rheumatic heart disease.

TABLE 1.—Types of Heart Disease in Pregnant Women

Type of Heart Disease	Pregnancies	
	No.	%
Rheumatic	395	83
Congenital	66	14
Hypertensive	6	1
Other	9	2
Total	476	100

TABLE 2.—Predominant Valve Lesion in Pregnant Patients with Rheumatic Heart Disease

Predominant Valve Lesion	Pregnancies	
	No.	%
Mitral stenosis	259	65
Mitral regurgitation	94	24
Aortic stenosis	3	1
Aortic regurgitation	39	10
Total	395	100

Table 2 presents the predominant valve lesion in patients with rheumatic heart disease. Two thirds of the pregnancies in women with rheumatic heart disease occurred in patients with predominant mitral stenosis.

TABLE 3.—Functional Classifications and Maternal Mortality in Patients with Rheumatic Heart Disease

Functional Class	Number of Pregnancies	Number of Maternal Deaths
I	212	0
II	85	0
III	32	1
IV	66	3
Total	395	4

In Table 3, the functional classification of the patients in each pregnancy is presented, along with the maternal mortality. The total maternal mortality in women with rheumatic heart disease amounts to 1%. No maternal deaths have occurred in women with other forms of heart disease, so that the total mortality in our series amounts to less than 1%.

TABLE 4.—Mortality of the Fetus and Newborn According to the Severity of Maternal Heart Disease

Maternal Functional Class	Number of Pregnancies	Number of Fetal and Neonatal Deaths		
		Spontaneous	Interruptions	Total %
I	212	11	2	6
II	85	6	5	13
III	32	2	7	28
IV	66	13	18	47
Total	395	32	32	16 (av.)

Table 4 presents the fetal and neonatal mortality rate according to cardiac classification of the mother. In general, increasing severity of maternal heart disease is associated with increasing fetal and neonatal mortality. This generalization is true even when the fetal deaths due to interruption of pregnancy for therapeutic reasons are excluded from the series.

A maternal mortality rate of 1% is not unique in our experience. It has been achieved in several large reporting clinics. It has occurred before the advent of heart surgery and it has been

TABLE 5.—Incidence of Therapeutic Abortion in Women with Heart Disease

Year	Number of Pregnancies	Interruptions	
		Number	%
1950*	27	5	18
1951	60	10	17
1952	46	6	13
1953	45	3	7
1954	65	3	5
1955	34	0	0
1956	42	2	5
1957	33	1	3
1958	46	2	4

* Based on six months.

achieved in the face of a falling use of interruption of pregnancy as a therapeutic tool. Table 5 shows the decline in interruption rate among pregnant women with heart disease at the Boston Lying-in Hospital. The fact seems clear that more women with heart disease are having more babies with greater safety than ever before.

Consideration of the effect of pregnancy on maternal heart disease should include an evaluation of its effect on the long-term prognosis of women who survive pregnancy. In general, reported studies (8) agree that no demonstrable deleterious effect on the course of heart disease results from successful pregnancy. A recent report by Donzelot and co-workers (13) from Paris is less optimistic, concluding that 2 of every 3 women manifest increased cardiac disability within 5 years following delivery.

Little information is presently available regarding the effects

of maternal heart disease on the health and development of their children. Such a study is currently in progress at the Boston Lying-in Hospital. In general, it may be said that except for congenital defects of the cardiovascular system the presence of heart disease in the mother does not appear to increase the risk of heart disease or other congenital malformations in the child.

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